

# HORSE MANAGENTENT HEALTH



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# HORSE MANAGEMENT: HEALTH

#### INTRODUCTION

As a horse owner you are often the person most familiar with the well-being of your horse and as such have the best understanding, by observation, of what is normal in your horse. Because of this, you would also be the first to see what is abnormal. Horse owners should become conscious of maintaining a healthy horse through preventive measures and by early recognition of disease signs. It is

important that the horse owner know when to seek professional advice about a problem, as best results are often based on early diagnosis. The more knowledgeable the horse owner becomes, the more appreciative of veterinary services he or she will be, as horse health problems can be very complex and difficult to pinpoint.

#### THE NORMAL HORSE

The normal healthy horse is alert, with the eyes wide open and the ears going to and fro. The lining of the eyes and nostrils are a salmon pink color and the horse has a good appetite and eats well. The coat is smooth and glossy and is easily moved over the ribs. The horse stands evenly on all four feet; a hind foot may be rested alternately but never a fore foot. Droppings are passed about eight times a day and are usually formed into a ball which breaks on hitting the ground. Urine, which is thick and yellow in color, is passed several times a day without difficulty. The normal temperature is 100.5°F (38°C) and the resting respiratory rate is about 10 to 12 a minute.

For a detailed inspection of a horse, first stand back and gain an overall, undisturbed impression. The horse should be responsive to his surroundings and show this with his eyes, ears and posture. Ask yourself whether the horse appears listless. Does he notice movements or noises around him? Is he standing off by himself? Extreme excitement may indicate severe pain such as colic. Look for obvious injuries, wounds, scrapes or types of unsoundness. Abnormal posture when the horse is standing, such as arching the back, indicates pain or discomfort. A horse that is rolling, kicking at his belly, sweating or lying down in an abnormal way may be in severe pain.

The hair coat and skin are often indicators of problems. During the summer, the hair coat should be bright and sleek with a gloss from the natural body oils. The winter hair coat should be thick, uniform and although not as bright, a glossy tinge should be apparent. Patches of missing hair or a dull, dry hair coat are worthy of concern. The skin, seen by parting the hair coat, should not be excessively dry or scaly.

It is important to observe any unusual eating habits. The horse may appear nervous or seize his food in an abnormal manner. When chewing, the horse may drop food from his mouth (called quidding) or stop and start chewing at various intervals for no apparent reason. Quidding is often due to teeth problems whereas the latter may be the

"dummy syndrome" due to brain damage. The horse may salivate excessively or have trouble swallowing.

The horse's manure should be watched on a regular basis for any consistency and/or color changes. Try to observe the horse defecating or urinating as any abnormality may indicate a problem.

Next, observe the fleshiness of the horse to determine if the horse is deficient in muscle or excessively thin or fat. If it is deficient in muscle, this will be most obvious in the hip and back as these areas are where the large muscles are located. A horse can be protein deficient and still have excess fat. Horses are overfat as a result of excess energy in the diet. Fat will cover the ribs and generally round out the natural contours of the body. Excess fat will accumulate between the back legs to give the hip a square look. Fat is sometimes mistaken for muscle and many show horses carry too much fat in order to give a false impression of muscle. A rough guide to determine the right amount of fat carried by the horse is that the ribs are easily felt but not seen, or fat should not exceed 6 mm in thickness. Emaciation is usually the result of an energy deficient diet, resulting in a loss of body fat and muscle making the skeletal structure very prominent.

Sunken eyeballs or excessively tight skin may indicate the horse has not been receiving or retaining sufficient water. Dehydration in the presence of adequate drinking water is a sign of severe disease.

One can now start a closer observation of the various body regions. Check the nostrils, eyes, tongue, teeth, jaws and ears. The eyes should be clear, bright, and free of debris and blemishes. To see if the horse has sight in both eyes, move your hand slowly towards each eye and observe if the horse follows the movements on both sides. The nostrils should be large, clear and slightly moist. Observe that both nostrils are free of discharge or any unusual odor. Abnormal odor may indicate dental or digestive problems. Open the horse's mouth to observe that his tongue is normal and

that the gums are moist and pale pink in color. Next check the teeth for abnormal wear, sharp edges or high spots. The molars develop sharp edges on the cheek side on the top and on the tongue side on the bottom. The rough edges. caused by abnormal growth and wear patterns can damage the cheeks or tongue. This is corrected by floating the teeth and is best done by an experienced person. The head should be observed for symmetry; any deviation may indicate nerve paralysis, fractured bones, or swellings due to infections or tumors. The throat latch area should be felt for enlargements. Now move to the chest and begin by trying to determine the number of heart beats per minute (pulse). The pulse can be taken directly over the heart just behind the elbow with a hand or a stethoscope. At rest, the normal rate is 30 to 40 beats per minute in the adult horse. Young horses have higher levels (a 2 week old foal may have a heart rate of 100). Resting pulse rates vary considerably depending upon the conditioning of the horse and the degree of excitement. An excessively high pulse rate (greater than 70 in the adult) may indicate extreme stress (pain, excitement, overwork, illness) whereas a very low pulse may indicate heart problems.

The respiration rate is the number of times the horse breathes per minute. One in and out movement, as observed in the flank region, is tallied as one count. The normal

resting respiration rate of the horse is 10 - 12 inhalations per minute. Resting respiration rates (as well as pulse rates) tend to decrease with conditioning. Rates greater than 40 may indicate fatigue. Respiration rates which are greater than pulse rates could indicate exhaustion. As with pulse rates considerable variation occurs between horses. One should also observe the rhythm and depth of respiration for abnormalities.

Droppings should be observed routinely. The fecal balls should be moist but not watery. Color will vary from browny green to light green depending on the diet. Abnormalities would include such things as being excessively dry or moist, having signs of blood, being extremely dark or containing worms. Horse owners should be constantly aware of the horse's diet, including the quality and quantity of feed and water consumed.

It is important to check the external genitalia of horses for abnormalities. These might include swellings or abnormal secretions (blood, pus, etc). The presence of only one testicle at puberty (about 12 months of age) is undesirable and a veterinarian should be consulted to geld the colt.

The limbs should be compared for symmetry. Visible limb abnormalities may include enlarged or distorted bones, joints, tendons or tendon sheaths.

#### **INTERNAL PARASITES OF HORSES**

Worms are the most common health problem in Alberta. A horse becomes infected, in most cases, by ingesting worm eggs or larvae that have passed out in its own manure or the manure of another horse. Consequently, anytime you have horses eating in the same areas in which they are defecating, the horses will invariably be infested with worms.

The damage caused by worms depends upon many factors, e.g., type of worm carried, the number of worms carried, the age of the horse, and concurrent stress such as poor nutrition, vigorous exercise, pregnancy, climate, disease. Often horses will cary a large worm burden and still

appear fat and healthy while being a source of infestation for clean horses.

There are several different types of worm which affect horses. Each has a specific life cycle which allows it to continue the species. A knowledge of these life cycles allows the horse owner to break the cycle at certain critical points, thereby inhibiting propagation of the species in question. For this reason a brief discussion of the various types of worms commonly found in Alberta and their life cycles will be included here, followed by methods of control and treatment.

# **TYPES OF WORMS**

# STRONGYLES: (Bloodworms)

These parasites are small, in the 1-5 cm range and they are commonly referred to as bloodworms or redworms. They are divided into large Strongyles and small Strongyles.

Under large Strongyles we find one of the most common and serious of the internal parasites of horses in Alberta, *Strongylus vulgaris*.

The eggs of Strongylus vulgaris are passed in the

manure of horses that are carrying the adult worms in their large intestines. The egg hatches in the manure to release a larva, or small immature worm. The larva is eaten by the horse while ground feeding or grazing. Inside the small intestine the larva sheds its outer layer and penetrates the wall of the intestine to enter an artery. Fourteen days after the larva has entered the horse it is found in the main artery supplying blood to the intestines. For three to four months this larva then migrates and wanders in the artery causing extensive damage which can result in impared circulation

of blood to the intestine. Eventually the larvae migrate back towards the intestine. After six to seven months these larvae have developed into adult worms and are found in the cecum and large intestine. As adult worms they are now capable of producing eggs to infect other horses or to reinfest their current host.

Severe colic and even death can be directly attributed to the damage done by these worms while migrating. This, plus the fact that most dewormers do not effectively kill the migrating larvae, makes this worm a serious problem. Most dewormers do effectively destroy adult Strongyles.

Other large Strongyles include *Strongylus edentatus* and *Strongylus equinus*. These do not migrate in blood vessels but can cause serious damage when present in high numbers.

Small Strongyles do not usually cause serious problems in adult horses.

The eggs of Strongyles can last through the winter and become infective in large numbers with the first thaw of spring. Eggs passed from March to October generally become infective within a few days.

# **ASCARIDS: (Roundworms)**

Ascarids are commonly referred to as roundworms and Parascaris equorum is the round worm of horses. This parasite is generally only of concern in horses under two years of age. This worm is very large, reaching a length of up to 50 cm. Eggs of this parasite are passed in the manure and can remain in the soil for several years. Eggs are eaten by the horse while it is ground feeding or grazing. The eggs hatch to larvae in the intestine. The larvae then migrate through the intestinal wall to the liver and diaphragm to reach the lungs. The time from ingestion of the egg until the larvae reach the lungs is 10-14 days. In the lungs the larvae migrate into the larger airways, then up the trachea to the throat and mouth where they are then swallowed returning them to the small intestine. The time it takes to arrive back in the intestine is about four weeks. The larvae develop into adults in the small intestine with females producing up to 200,000 eggs per day.

Ascarid larvae can cause damage to the liver and lungs of horses if they migrate in large numbers. They can also leave these organs susceptible to bacterial infections. As adults, ascarids are big enough to block the intestines of young horses when the worms are present in large numbers.

# BOTS

There are three very common species of bots which affect horses and all have a similar life cycle. The bot is really not a worm but the larval stage of the gastrophilus fly. Starting in July and ending with the first killing frost, the bot

flies lay their yellow-colored eggs around the horse's head, chest or front legs. When the eggs become warm and moist they hatch and the larvae find their way to the horse's mouth (often through the horse's licking). The larvae migrate through the mouth for a couple of weeks, sometimes causing the tongue and gums to swell slightly.

Eventually they are swallowed and develop into larger larvae, of a more advanced stage, which attach themselves to the wall of the stomach. These remain in the stomach, for a variable period, often over winter, until they pass out in the feces, especially in the spring, pupate and develop into adult flies. Horses affected with bots may suffer from loss of condition. Where the infestation is severe, bots can cause colic by obstruction of the stomach and/or duodenum (first portion of the intestine). It is possible for these parasites to destroy the mucous lining of the stomach or to penetrate the stomach wall. Adult bot flies have underdeveloped mouth parts and do not bite as is commonly believed.

Bot larvae are not usually killed by routine horse wormers. Treatment specifically for bots should be used every fall after the first hard frost. Removing the eggs from the legs of horses, when they are caught up during the summer, will lessen the number of bots that will be in the stomach.

# **OXYURIS EQUI: (Pinworms)**

Oxyuris equi is the name given to the common worm which lives in the large intestine of horses. The female worm migrates to the anus to lay her eggs, depositing them around the anus. This causes intense itching in this area and affected horses will often rub their tails against fences, buildings, etc. Infestation occurs by ingestion of eggs. Other than the discomfort caused during egg-laying these worms have little harmful effect on the horse.

# **THREADWORMS**

Strongyloides westeri is a small threadlike worm which inhabits the small intestine as an adult. Only females live a parasitic existence and they are able to produce eggs asexually which pass out in the feces. The larvae which hatch from these eggs can infest horses directly or can develop into adult males and females which then reproduce sexually. The larvae live on the pasture until they are ingested by a horse, or die from environmental exposure. Infestation of horses may occur by ingestion during grazing, through the mare's milk (they appear as early as four days after foaling) or by penetrating the skin, whereupon they enter blood vessels and migrate to the liver, lungs, and up the trachea to be swallowed and thus establish themselves as adults in the small intestine. Appreciable numbers of these worms are

usually found in young foals only, as older horses develop a resistance to them. Affected foals may have slight diarrhea but severe problems caused by this parasite are rare.

#### STOMACH WORMS

Habronema megastoma is a relatively small (1 cm) worm which lives in the stomach of the horse. Eggs pass out in the feces, then the larvae hatch and infect the developing maggots of horse and stable flies. These maggots develop into adult flies which still carry the worm larvae. Horses become infected by ingesting an affected fly or maggot. Flies may also deposit the worm larvae in open cuts, causing a condition known as summer sores. These sores are very difficult to heal. The larvae which reach the horse's stomach develop into adult worms which may cause a mild to severe stomach upset with a very rapid loss of condition.

# **TAPEWORMS**

There are three species of tapeworm which affect horses. These require a small pasture mite as an intermediate host. Horses become infested by ingesting infected mites. Tapeworm infections are not common in Alberta and light infections usually do not pose a problem for the horse.

Intestinal obstruction may occur in rare instances of heavy tapeworm infestation.

#### LUNGWORMS

The lungworm which horses can get is *Dictyocaulus arnfieldi*. This is more common in donkeys and mules. Horses usually get lungworms only when they are pastured or housed with donkeys or mules. The egg of the lungworm is passed by the donkey or mule in the manure and in 3 to 14 days hatches to a larva. The larva is eaten by the horse and goes to the small intestine. Here it burrows in the wall of the intestine and enters the lymphatic circulation which carries it to the lungs. Infested horses have a persistent cough and only rarely pass eggs to infect other horses. Infested donkeys show no signs of illness and regularly pass eggs of the parasite.

The manure of donkeys should be checked regularly for lungworm. The manure must be very fresh for examination, as a special test known as a Baerman Test is required. Horses pastured with donkeys that are positive for lungworm should be treated. Donkeys and mules should be treated regularly if positive. A veterinarian should be consulted as to which dewormer to use as very few are effective against lungworm.

#### **CONTROL OF INTERNAL PARASITES IN HORSES**

Although prevention of worm infections would be ideal, it is probably impossible under practical management conditions. For this reason, controlling worm infections at a level compatible with the economic and management limitations of each individual horse enterprise is the aim.

The main point to consider in controlling worm infection is to limit exposure of the horse to infective eggs and larvae and to effectively treat horses known to be carrying an excessive parasite burden. There are several management techniques which will aid in limiting exposure of a horse to infective eggs and larvae:

Avoid overcrowding in pastures.

Practise rotation grazing. Keeping horses off a contaminated pasture for an extended period (one year) will allow the pasture to reduce its contamination through environmental destruction of most of the eggs and larvae. (*Parascaris equorum* eggs are very resistant and may survive).

Practise mixed grazing; eggs or larvae of horse worms that are ingested by cows or sheep, etc. will not develop, but the eggs and larvae are effectively removed from the pasture.

Plowing and reseeding of pastures, where feasible, is a very effective method of decontamination. Plowing to a depth of 23 cm allows for efficient burying of eggs and larvae on the pasture.

Never give supplemental feed on the ground as this forces horses to eat in the same areas in which they defecate. Properly constructed feed bunks and troughs should

be used in place of ground feeding.

Never spread horse manure from barns and stalls on land intended for horse pasture. Properly composting the manure (one year) will result in destruction of the parasites after which it may be safely spread on the pasture.

Never turn affected horses out on a clean pasture. Fecal examination for eggs can identify affected horses, which must then be dewormed before being turned out onto new pasture.

Avoid overgrazing. There is a tight "mat" of dead plant material, humus, etc. which is very close to the ground surface. This mat provides parasitic larvae and eggs with protection from desiccation by sunlight and is therefore a rich source of infestation for horses which may be forced by graze very close.

In stables, follow strict sanitation measures. Clean waterers, feed troughs, hay bunks and stalls frequently and properly.

Picking up manure on pastures is an effective, although somewhat tedious method of preventing pasture contamination. To be effective this should be done frequently, as time and labor permit. The manure can then be composted before returning it to the pasture.

Chain harrowing of pastures may be effective in that it breaks up the manure piles and thus exposes the parasites (especially strongyle larvae) to desiccation by the sun. Since this method also tends to spread the parasites over the entire pasture, horses should not graze on it for about

a month after harrowing so that the dessication process is complete.

Frequent clipping or washing of the front legs and beneath the lower jaw with warm water to remove bot eggs is an effective method of reducing bot infestation during the

fly season.

Regular deworming treatment kills egg-laying adults in the intestine and greatly reduces pasture contamination.

Do not let horses drink stagnant water or water which drains from areas contaminated by manure.

#### TREATMENT OF INTERNAL PARASITES

One of the most effective methods of breaking a worm's life cycle and thus controlling the worm population is regular treatment with anthelmintics (dewormers). This practice inhibits the physical damage caused by adult worms in the gastrointestinal tract and decreases pasture contamination by removal of the egg source. An effective worm control program should be set up in consultation with a veterinarian. Fecal examination for eggs and larvae is a simple and effective means which may be used by the veterinarian to determine if a worm problem is present in horses.

The young horse is most susceptible to the adverse effects of worm parasitism and early care is necessary to prevent irreversible damage being done by a heavy worm load. Foals should be first dewormed at two months of age if affected, and a veterinarian should be consulted about frequency of subsequent treatments. Older horses act as reservoirs of infection for young horses, as well as being adversely affected by a heavy worm load themselves. Therefore, it is important to include them in a worm control

Anthelmintics may be administered in one of four ways. Some effective preparations can be added to the feed. This method is often unsuccessful as some horses refuse to eat the medicated feed or "eat around" the dewormer. A more effective method of deworming is with the stomach tube. However, let a veterinarian do it as the risk of forcing fluid into the lungs is high. Drenching is another method of anthelmintic application. Its use is to be discouraged because of the risk of the drench entering the lungs if the horse does not swallow. Newer paste type preparations are effective and safe. Continuous feeding of low levels of certain anthelmintics has been shown to be a very effective and cheap method of controlling worms, especially roundworms. If your management practice will allow this type of program, discuss the details with your veterinarian.

Table 1 compares the effectiveness of different preparations. Consult your veterinarian about the choice of anthelmintic best suited to your purpose. Often less expensive, feed type preparations contain a dewormer that is effective against only certain types of worms and are not necessarily effective against all types of worms affecting your horse. During the summer, use preparations that are effective against strongyles, ascarids, and pinworms. During the winter use one that is effective against bots as well as the above three worms.

Table 1 - Efficacy of Antinarasitic Compounds Used in Horses

Carbon Disulfide (CDS)         None         .05 ml/kg         T         90-100         50-100         0         0         0         0         0         0         Phenothiazine (PTZ)         Various         4.4 (LL)         F         0         0         95-100         95-100         95-100         0         0         Phenothiazine (PTZ)         Various         55         T,F,P         0         0         95-100         95-100         95-100         0         0         0         0         Phenothiazine (PTZ)         Various         55         T,F,P         0         0         50-75         20-40         85-95         0         25-50         Piperazine (PPZ)         Various         88* 78         T         75-85         95-100         40-60         0-10         90-100         40-60         0-15         Piperazine (PPZ)         Various         28+88         LT         0         95-100         40-60         0-10         90-100         50-70         10-20         90-100         70-70         10-20         90-100         70-70         10-20         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90		Average Removal Expectancy %										
Carbon Disulfiide (CDS)         None         .05 mi/kg         T         90-100         50-100         0         0         0         0           Phenothiazine (PTZ)         Various         4.4 (LL)         F         0         0         95-100         95-100         0         0           Phenothiazine (PTZ)         Various         55         T.F.P         0         0         95-100         95-100         0         0           Piperazine (PPZ)         Various         88*         T         0         95-100         40-60         0-10         90-100         40-60         0-15           Picadex (PPZ-CDS)         Parex         88**78         T         75-85         95-100         40-60         0-10         90-100         50-70         10-20           PTZ + PPZ Various         28+88         LT         0         95-100         90-100         70-90         90-100         50-70         20-40           PTZ + PPZ CDS         Parex Plus         0.65 ml/kg         T         65-75         95-100         90-100         70-90         90-100         50-70         20-40           PTZ + PPZ - CDS         Parex Plus         0.65 ml/kg         T         65-75         95-100         90-100	Compound		Dose			Strongyles		Oxyurids		Toxicosis		
Phenothiazine (PTZ)   Various   4.4 (LL)   F   0   0   0   95-100   95-100   95-100   0   0	Generic Name	Trade Name	mg/kg	Method	Bots	Ascarids	S. vulgaris	S. edentatus	Small	Mature	Immature	factor
Phenothiazine (PTZ)   Various   55   T.F.P.   0   0   50-75   20-40   85-95   0   25-50	Carbon Disulfide (CDS)	None	.05 ml/kg	T	90-100	50-100	0	0	0	0	0	1X
Piperazine (PPZ)   Various   88"   T	Phenothiazine (PTZ)	Various	4.4 (LL)	F	0	0	95-100	95-100	95-100	0	0	-
Picadex (PPZ-CDS)         Parvex         88*-78         T         75-85         95-100         40-60         0-10         90-100         50-70         10-20           PTZ + PPZ - CDS         Parwex Plus         0.65 ml/kg         T         65-75         95-100         90-100         40-60         90-100         50-70         20-40           PTZ + PPZ-CDS         Parwex Plus         0.65 ml/kg         T         65-75         95-100         90-100         70-90         90-100         50-70         20-40           Trichlorfor (TCF)         Dyrex Cap Tabs         80         B         95-100         96-100         0         0         0         90-100         90-100         N.D.           PTZ + PPZ + TCF         Dyrex, T.F.         28+88*40         T         90-100         95-100         30-50         90-100 </td <td>Phenothiazine (PTZ)</td> <td>Various</td> <td>55</td> <td>T,F,P</td> <td>0</td> <td>0</td> <td>50-75</td> <td>20-40</td> <td>85-95</td> <td>0</td> <td>25-50</td> <td>1X</td>	Phenothiazine (PTZ)	Various	55	T,F,P	0	0	50-75	20-40	85-95	0	25-50	1X
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Piperazine (PPZ)	Various	88*	T	0	95-100	40-60	0-10	90-100	40-60	0-15	3X
PTZ + PPZ-CDS Parvex Plus 0.65 ml/kg T 65-75 95-100 90-10 70-90 90-10 70-90 90-100 50-70 20-40 PTrichlorfon (TCF) Dyrex Cap Tabs 80 8 95-100 95-100 90-100 70-80 90-100 90-100 90-100 N.D. PTZ + PPZ + TCF Dyrex, T.F. 28+88*40 T 90-100 95-100 95-100 95-100 95-100 90-100	Picadex (PPZ-CDS)	Parvex	88*+78	T	75-85	95-100	40-60	0-10	90-100	50-70	10-20	3
Trichlorfon (TCF)         Dyrex, ComBot         40         T,F,P         90-100         95-100         0         0         0         90-100         N.D.           Trichlorfon (TCF)         Dyrex Cap Tabs         80         B         95-100         95-100         90-100         70-80         90-100<	PTZ + PPZ	Various	28+88	LT	0	95-100	90-100	40-60	90-100	50-70	20-40	2XI
Trichlorfon (TCF)         Dyrex Cap Tabs         80         B         95-100         95-100         90-100         70-80         90-100         90-100         N.D           PTZ + PPZ + TCF         Dyrex, T.F.         28+88*40         T         90-100         95-100         95-100         30-50         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         90-100         30-40         10-20         10-20         95-100         95-100         90-100         90-100         90-100         30-40         30-40         10-20         10-20         95-100         95-100         90-100         90-100         90-100         30-40         30-40         10-20         10-20         95-100         95-100         90-100         90-100         90-100         30-40         90-100 </td <td>PTZ + PPZ-CDS</td> <td>Parvex Plus</td> <td>0.65 ml/kg</td> <td>T</td> <td>65-75</td> <td>95-100</td> <td>90-10</td> <td>70-90</td> <td>90-100</td> <td>50-70</td> <td>20-40</td> <td>4X</td>	PTZ + PPZ-CDS	Parvex Plus	0.65 ml/kg	T	65-75	95-100	90-10	70-90	90-100	50-70	20-40	4X
Trichiorfon (TCF)	Trichlorfon (TCF)	Dyrex, ComBot	40	T.F.P	90-100	95-100	0	0	0	90-100	N.D.	1X
Thiabendazole (TBZ)	Trichlorfon (TCF)		80	В	95-100	95-100	90-100	70-80	90-100	90-100	N.D.	1X
Thiabendazole (TBZ)         Equizole e (44-88**         T,F,P         0         10-75         95-100         90-100         90-100         90-100         30-40           TBX + PPZ         Equizole A         44+55*         T,F         0         10-75         95-100         90-100         90-100         90-100         30-40           TBZ + TCF         Equizole B         44+40         T,F         95-100         95-100         90-100         90-100         90-100         30-40           Dichlorvos (DDVP)         Equigard         35         F         80-100         95-100         95-100         70-100         85-95         90-100         90-100           Dichlorvos (DDVP)         Equigel         10-20**         P         90-100         99-100         0-10         N.D.         90-100         90-100           Pyrantel pamoate         Strongid T, Pyraminth 66*         T,F,S         0         90-100         995-100         65-75         90-100         95-100           MBZ + TCF         Telmin, S,F.         *8.8         T,F,P         0         95-100         95-100         65-95         80-95         95-100         95-100           Cambendazole (MBZ)         Tilmin B         8.8+40         LT,F         95-100 </td <td>PTZ + PPZ + TCF</td> <td>Dyrex, T.F.</td> <td>28+88*+40</td> <td>T</td> <td>90-100</td> <td>95-100</td> <td>95-100</td> <td>30-50</td> <td>90-100</td> <td>90-100</td> <td>90-100</td> <td>1X</td>	PTZ + PPZ + TCF	Dyrex, T.F.	28+88*+40	T	90-100	95-100	95-100	30-50	90-100	90-100	90-100	1X
TBZ + TCF	Thiabendazole (TBZ)		°44-88**	T,F,P	0	10-75	95-100	90-100	90-100	90-100	30-40	25X
Dichloros (DDVP)         Equigard         35         F         80-100         95-100         95-100         70-100         85-95         90-100         90-100           Dichloros (DDVP)         Equigal         10-20**         P         90-100         90-100         0-10         0         N.D.         90-100         90-100           Pyrantel pamoate         Strongia T, Pyraminth         66*         T,F,S         0         90-100         995-100         65-75         90-100         670         50-60           Mebendazole (MBZ)         Telmin, S,F.         *8.8         T,F,P         0         95-100         90-100         65-95         80-95         95-100         95-100           Pambendazole (CBZ)         Camwet         20         T,F,P         0         95-100	TBX + PPZ	Equizole A	44+55*	T,F	0	95-100	95-100	90-100	90-100	90-100	30-40	1X
Dichloryos (DDVP)         Equigel         10-20**         P         90-100         90-100         0-10         0         N.D.         90-100         9.D         90-100         90-100         90-100         0-10         N.D.         90-100         90-100         50-60           Mebendazole (MBZ)         Telmin, S.F.         *8.8         T.F.P.         0         95-100         99-100         65-95         80-95         95-100         95-100           MBZ+ TCF         Telmin B         8.84-0         L.T.F.         95-100         95-100         65-95         80-95         95-100         95-100           Cambendazole (CBZ)         Camwet         20         T.F.P.         0         95-100	TBZ + TCF	Equizole B	44+40	T,F	95-100	95-100	95-100	90-100	90-100	90-100	30-40	1 X
Dichloryos (DDVP)         Equigel         10-20**         P         90-100         90-100         0-10         0         N.D.         90-100         9.D         90-100         90-100         90-100         0-10         N.D.         90-100         90-100         50-60           Mebendazole (MBZ)         Telmin, S.F.         *8.8         T.F.P.         0         95-100         99-100         65-95         80-95         95-100         95-100           MBZ+ TCF         Telmin B         8.84-0         L.T.F.         95-100         95-100         65-95         80-95         95-100         95-100           Cambendazole (CBZ)         Camwet         20         T.F.P.         0         95-100	Dichlorvos (DDVP)	Equigard	35	F	80-100	95-100	95-100	70-100	85-95	90-100	90-100	3X
Mebendazole (MBZ)         Telmin S.F.         *8.8         T.F.P.         0         95-100         90-100         65-95         80-95         95-100         95-100           MBZ+TCF         Telmin B         8.8+40         LT,F         95-100         95-100         95-100         65-95         80-95         95-100         95-100           Cambendazole (CBZ)         Camwet         20         T.F.P         0         95-100         95-100         90-100         95-100 <td></td> <td></td> <td>10-20**</td> <td>Р</td> <td>90-100</td> <td>90-100</td> <td>0-10</td> <td>0</td> <td>N.D.</td> <td>90-100</td> <td>N.D.</td> <td>2X</td>			10-20**	Р	90-100	90-100	0-10	0	N.D.	90-100	N.D.	2X
MBZ + TCF         Telmin B         8.8+40         LT,F         95-100         95-100         95-100         65-95         80-95         95-100         95-100           Cambendazole (CBZ)         Camvet         20         T,F,P         0         95-100         95-100         90-100         95-100 <td>Pyrantel pamoate</td> <td>Strongid T, Pyraminth</td> <td>6.6"</td> <td>T,F,S</td> <td>0</td> <td>90-100</td> <td>995-100</td> <td>65-75</td> <td>90-100</td> <td>60-70</td> <td>50-60</td> <td>20X ET</td>	Pyrantel pamoate	Strongid T, Pyraminth	6.6"	T,F,S	0	90-100	995-100	65-75	90-100	60-70	50-60	20X ET
Cambendazole (CBZ)         Camvet         20         T,F,P         0         95-10	Mebendazole (MBZ)	Telmin, S.F.	°8.8	T,F,P	0	95-100	90-100	65-95	80-95	95-100	95-100	40X ET
Fenbendacole (BVZ)         Panacur         5-10**         T.F.D         0         90-100         95-100	MBZ + TCF	Telmin B	8.8+40	LT,F	95-100	95-100	95-100	65-95	80-95	95-100	95-100	1X
Febantel (FBT)         Rintal         6         T.F.P.         0         95-100 <td>Cambendazole (CBZ)</td> <td>Camvet</td> <td>20</td> <td>T,F,P</td> <td>0</td> <td>95-100</td> <td>95-100</td> <td>95-100</td> <td>90-100</td> <td>95-100</td> <td>95-100</td> <td>30X ET</td>	Cambendazole (CBZ)	Camvet	20	T,F,P	0	95-100	95-100	95-100	90-100	95-100	95-100	30X ET
Oxfendazole (OFZ) Benzelmin °10 T,F 0 90-100 95-100 95-100 95-100 95-100	Fenbendazole (BVZ)	Panacur	5-10**	T,F,D	0	90-100	95-100	90-100	95-100	95-100	50	100X
	Febantel (FBT)	Rintal	6	T,F,P	0	95-100	95-100	95-100	95-100	95-100	95-100	40X
	Oxfendazole (OFZ)	Benzelmin	° 10		0	90-100	95-100	95-100	95-100	9500	95-100	10X
Oxibendazole (OBZ) Anthelcide-EQ 10 T.F 0 90-100 95-100 95-100 95-100 95-100	Oxibendazole (OBZ)	Anthelcide-EQ	10	T,F	0	90-100	95-100	95-100	95-100	95-100	95-100	60X

<sup>=</sup> Values for susceptible populations; drug resistance may be encountered,

Reference; Adapted from: Drudge J.H. et al: Parasite Control in Horses: A Summary of Contemporary Drugs Veterinary Medicine/Small Animal Clinician. Oct 1981: 1479-1489.

especially among strongyles.

base content

<sup>\*\*</sup> higher dose rate for ascarids and/or pinworms

O Not effective against Strongyloides westeri at these dosages

ET - effective against tapeworms

effective against immature stages

<sup>+</sup> effective against microfilaria of *Onchorerca cerviacalis*LL - Low level system - 2.0 g/horse/day, 21 days each month
T - stomach tube, F - feed, B - Bolus, P - Paste or Gel, D - Drench, S - dosing syringe

N.D. - Not determined Toxicosis factor = Safe within - X the recommended dose

#### LICE

Horses can be infested by either sucking or biting lice. These lice are specific to horses and do not infest cattle. Long winter coats are ideal for the build up and spread of lice, so infestation is more common in winter. The life cycle of lice involves the adult laying eggs (nits) on the horse's hair. These nits incubate for 5 to 14 days, then hatch into nymphs. Nymphs feed and develop into adults in two weeks. Adults continue feeding and lay 50-100 eggs in their life span of 4 to 5 weeks. Spread is dependent on animal contact and therefore crowding is an important factor. Contaminated grooming equipment or tack may also be a source of infestation. Horses being brought in should be checked for lice. Lice occur on all areas of the body but owing to the animal's self grooming are usually first noticed on the head, neck, mane and tail. An inexperienced person may have trouble seeing a louse infestation until it is quite severe. The horse becomes nervous and tends to bite and scratch excessively. During severe infestations a considerable loss of hair and a poor skin condition will be noticed.

The blood sucking louse can result in anemia, weakness and contribute to stunted growth and loss of condition. Treatment usually involves an insecticide applied to the entire body of the horse in either a liquid or powder form. Ivermectin, the popular anthelminthic, is also a very effective treatment for infested horses. Any equipment which may have been infested must also be treated to prevent reinfestation.

# WINTER TICKS

The winter tick, *Dermacentor alliepictus*, has a very wide distribution in North America. Unlike the other three host ticks, this tick only has one host during its life cycle. The larvae of these ticks attach themselves to the horse and feed on blood. They develop into adults in 5-14 weeks, mate and then the female drops to the ground to lay eggs. Egg laying can be delayed 4 to 5 months if females drop in early winter. Larvae hatch in one to two months and when fall comes they climb to the tips of grass and become attached to the horse as it walks through the grass. On horses with long winter hair the infestation often goes unnoticed until quite severe. Horses usually rub, scratch and bite exces-

sively. The infestation can result in weakness, emaciation, loss of appetite, anemia and even death to foals. Abdominal swelling called "water belly" can occur with severe infestations.

#### **FLIES**

Flies can become a serious problem to the horse owner during the summer. Horse flies can become a problem where excess waste material builds up. Besides being bothersome they can transmit stomach worms (Habronema). The stable fly (Stomoxys calcitrans) bites and sucks blood. Both the horses and their owners are susceptible to these attacks. Stable flies reproduce in horse manure and are also capable of transmitting the stomach worm. Horses may be attacked by the horn fly (Haematobia irritans) when pastured or working with cattle. These flies remain on the horse day and night around the head and neck, backline, abdomen and legs. The face fly (Musca autumnalis) is a pest of horses, cattle and sheep. It breeds in fresh cattle droppings and attacks horses kept on pasture with cattle. It feeds on eye, nose and mouth secretions as well as those from open wounds. The fly only remains on the horse long enough to feed. Horse and deer flies are bloodsuckers and a common problem of pastured horses.

Fly control depends on good manure management, water management and general sanitation. This involves manure removal and keeping stalls freshly bedded. Good drainage from paddocks and corrals is important in reducing fly (and mosquito) populations.

Sanitation involves the removal of dead animals and afterbirth to burying grounds. Any wounds should be cared for and fly repellents administered as directed. All garbage, spoiled or spilled feed, organic wastes and pet droppings should be removed from pasture and barns. Preventing weed overgrowth allows more air movement, aids in drying manure and reduces hiding places. There are many insecticides available for fly control. These can be used on the horse and its environment. Some precautions are necessary. One should not spray horses in an unventilated area. Foals less than three months old should not be done and foals between three to six months done lightly. Take care not to contaminate drinking water or feed with insecticides. Most commercial fly repellents have complete instructions.

#### VACCINATION AND DISEASE CONTROL

To understand how vaccines work, it is necessary to have an understanding of how the immune system works and the effect vaccines have on the immune system. It has been known for centuries that individuals who contracted and lived through a particular disease were often resistant

to contracting the same disease again. This phenomenon is attributable to the stimulation of the natural defence system of the body by the organism responsible for the disease, thus leaving the animal "sensitized" to that particular organism. Upon re-exposure to the same organism the animal is

able to mount a very fast and efficient immune response which destroys the invader before it can exert its harmful effect.

It is not known how the body is able to recognize a "foreign" invader which may be introduced into its domain nor how it subsequently mounts a specific response against that particular invader. It is known, however, that by altering potential invaders (by treating them with formalin, growing them on culture media, etc.) their antigenic properties (ability to cause a specific immune response) can be retained while their ability to cause clinical disease is destroyed. This is the basis for the action of vaccines. When the vaccinated individual is subsequently exposed to the unaltered, disease-causing organism, the body is already "primed" for the attack and is able to repel the organism before harm is done.

As would be expected, not all disease-causing organisms follow the above plan and many have eluded attempts to produce effective vaccines against them. Others may require various applications with regard to route, frequency of administration, age at first administration, condition at the time of administration, etc., in order to produce effective immunity without risk of unwanted side effects.

In Alberta there are several diseases for which vaccines are available. Some of these vaccines are safe and effective, others are less desirable and their use is limited. These diseases will be discussed with reference to their symptoms, treatment and control. New and improved vaccines are constantly being produced and your veterinarian is best qualified to give you the latest information on new developments.

# **TETANUS (LOCKJAW)**

Tetanus is caused by the bacterial agent *Clostridium tetani* which is very closely related to the organism responsible for causing blackleg in cattle. The tetanus causing organism produces spores which are very small, seed-like structures capable of surviving for years in contaminated soil etc. To cause the disease these spores must enter the horse's body by means of a wound. One of the most common wounds is a nail puncture of the foot. Once in the body the spores can grow to the bacterial form and begin producing a very powerful toxin which spreads throughout the body seriously interfering with nerve function.

Usually within three days to a month of infection of a contaminated wound, injection, etc. the affected horse will begin showing symptoms. The first signs are a reluctance to eat off the ground and protrusion of the third eyelid (a pink fleshy structure) over the eyeball. As the disease progresses muscle stiffness will be noted, progressing to muscle spasms in which the horse stands in a stiff rigid position with tail extended, head and ears erect. The horse may fall down and be unable to rise. In about 80 per cent of all cases the horse will die.

At first signs of tetanus the horse should be moved to a quiet darkened box stall and a veterinarian called immediately. It is extremely important that the horse not be excited if possible. The veterinarian can use a variety of medications to treat the horse and treatment can be reasonably successful if instituted early and followed by good care.

Vaccination is a very effective method of tetanus prevention. Mares should be vaccinated one to two months before foaling; this will protect the foal until it is eight to ten weeks old. At eight weeks of age vaccinate the foal and give a booster innoculation six to eight weeks later. Annual innoculations are recommended for all horses every two or three years. Vaccinated horses that receive cuts or penetrating injuries should be given a booster shot in addition to thorough cleaning and dressing of the wound.

# STRANGLES (DISTEMPER)

Strangles is a bacterial disease caused by an organism known as *Streptococcus equi*. Horses become infected through direct contact with infected horses, or food and water on premises contaminated by an infected horse. Contaminated premises will remain infective for a year or more. When strangles occurs in a herd of horses it can spread rapidly and may affect many others. Affected horses will usually recover but occasionally one dies or a condition known as bastard strangles will occur in which the infection spreads to many parts of the body. Proper treatment usually ensures successful recovery.

Affected horses will have an increased temperature, a nasal discharge and will refuse food and water. Abscesses of various sizes will occur between the jaws and/or below the ear as the disease progreses; these may break and drain pus.

Veterinary assistance should be obtained in the initial treatment and appropriate antibiotics will be prescribed. The application of poultices to the swellings will aid in drawing them to a head to allow for subsequent opening.

Effective control is achieved through good management. Always isolate new horses or horses thought to have been exposed for two weeks before introduction into the herd. Use STRICT SANITATION procedures to prevent contamination of your premises. Several vaccines are presently available. Some have serious side effects (i.e. abscessation at the injection site). It is probably best to consult your veterinarian prior to using these vaccines.

# EASTERN AND WESTERN EQUINE ENCEPHALOMYELITIS (Sleeping Sickness)

Sleeping sickness is a disease of the central nervous system caused by a mosquito borne virus. The virus can be

found in many types of animals, including birds, rodents and reptiles, from which mosquitoes carry the virus to horses and man. It seems that both man and the horse are "dead-end hosts" in that they do not act as sources of infection for each other or other animals. The virus can cause death in both man and members of the horse family. The virus which causes Western Equine Encephalitis (WEE) differs slightly from the virus causing Eastern Equine Encephalitis (EEE), the latter being more often fatal (90 per cent for EEE as opposed to 50 per cent for WEE). The WEE occurs periodically in Alberta.

Affected horses will show a variety of signs. One experienced veterinarian describes "sleepers, circlers and bangers" as the different types of symptoms seen. Marked depression, high fever, lack of coordination and a deteriorating condition are the consistent signs of the disease. There is no treatment that can be directed against the virus itself, however, good supportive therapy under the direction of your veterinarian can greatly improve the chances of recovery. Horses that recover may have permanent brain damage although complete recovery is possible with good supportive care.

Prevention of the infection can be established through yearly vaccination prior to the mosquito season (see table of recommendations).

# RABIES

Rabies is a viral disease important for its public health significance. This fatal disease is rare in both horses and man. The virus is present in salivary secretions of infected animals and is transmitted through bites. Bats, skunks and foxes are the natural reservoirs of infection. It is conceivable that a rabid horse could attack and bite a human. A horse owner could be exposed by reaching into the mouth of a horse having difficulty in swallowing (which may be a sign of rabies).

Equine rabies can be very difficult to diagnose because of the broad spectrum of clinical signs. Clinical signs observed in rabid horses include depression, hyperexcitability, tremors, spasms, lameness, difficulty in chewing and swallowing, and paralysis. As a rule affected horses will deteriorate rapidly and die within five days of the onset of clinical signs.

Vaccines are available, but because of the extremely low incidence in Alberta, they are not routinely administered. A veterinarian should be contacted in all suspected cases of rabies.

#### **INFLUENZA**

Influenza is a very common disease among horses which congregate at shows, rodeos, racetracks. It is caused

by a virus which has the ability to spread rapidly among horses in close contact. Infected horses will shed the virus for about one week after a three day incubation period.

Fever, an explosive dry cough and nasal discharge are the usual signs of influenza. There may be slight swelling between the jaws in the early stages. The untreated horse may develop pneumonia caused by secondary bacterial infection. The severity of the disease will vary from mild to fatal, depending on age, condition, and the presence of a secondary bacterial infection.

There is no treatment against the virus itself. However, because the virus lowers the horse's resistance, secondary bacterial respiratory infections may occur if the animal is not properly treated.

Vaccines are available which are quite effective in controlling influenza. They are recommended for horses that are moved around to shows, races, etc. or for horses that are exposed to a constant flux of new horses, such as would be encountered at a breeding farm or training stable. The vaccination regime will vary according to the individual situation.

# **EQUINE RHINOPNEUMONITIS (Equine viral abortion)**

Rhinopneumonitis is caused by a herpes virus similar to that which causes cold sores in man. It causes a mild respiratory tract inflammation very similar to the influenza virus; the main concern is that exposed susceptible pregnant mares may abort or give birth to weak foals. On infected farms "abortion storms" may occur, usually in early spring when the mares are in the later stages of gestation and without the mares showing any other signs of illness.

Young horses will show a mild, transient upper respiratory infection with fever, depression, coughing and perhaps a slight swelling between the jaws or in the throat region. Older horses often do not show overt signs when infected, but susceptible pregnant mares will abort, usually in the eighth to tenth month of pregnancy. It is recommended that one submit the freshly aborted fetus and placenta to a provincial pathology lab for a causative diagnosis.

There is no treatment that can be directed against the virus. Any treatment used would be to prevent a secondary bacterial infection from attacking the weakened horse.

Effective vaccines are now available to assist in preventing abortion in mares. These must be started early in the pregnancy and should not be regarded as totally effective. Do not place new horses with pregnant mares to prevent introduction of the virus to the mares. Also isolate any mare that aborts, then clean and disinfect the area where she aborted. Do not allow the other mares access to the aborted fetus or placenta. Do not return the mare to the herd for at least three weeks after the abortion. Consult your veterinarian as soon as the mare aborts to arrange for diagnosis of the cause of the abortion.

# **EQUINE INFECTIOUS ANEMIA (Swamp Fever)**

This disease is caused by a virus which has the unique capability of infecting the horse for life, producing intermittent attacks of illness (during which the horse may die) followed by periods during which the horse may appear normal. The disease is spread by any means that will transfer blood from one horse to another such as biting insects, contaminated syringes or contaminated surgical instruments. The virus can survive boiling for 15 minutes, therefore simply washing a needle and syringe or castrating device does not ensure decontamination.

Affected horses will show a variety of signs from appearing outwardly normal to death. Horses remain infested for life and can act as sources of infestation for other horses. During acute attacks affected horses have a fever, are weak and lose condition. Death can occur at any time. In

the chronic state horses usually do poorly, tire easily and have intermittent bouts of fever which may culminate in an acute, fatal attack.

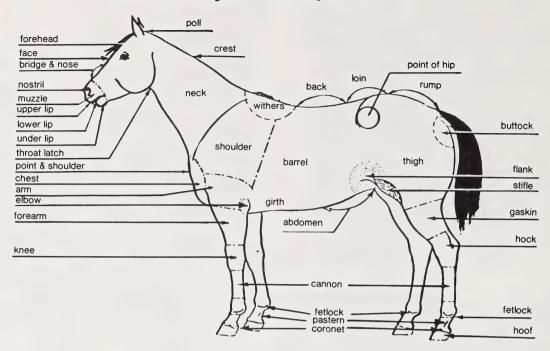
No treatment is currently available that will remove the virus from the horse.

There are no effective vaccines currently available. The Coggin's test will identify horses carrying the virus. By identifying and destroying horses testing positive, the source of infestation can be reduced. Managers of stables, tracks, training centers, breeding farms or any other facility with a constant flux of horses often require a negative Coggin's test before admitting any horse for safety reasons. It is a simple, accurate test performed on a blood sample collected by an approved veterinarian and sent to the federal laboratory. Horses that show a positive test must be reported to the federal health of animals branch, and by law be destroyed or totally isolated.

Table 2. - IMMUNIZATION PROGRAMS FOR HORSES

Disease	Recommendations for Immunization	Initial Immunization	Annual Boosters
Western & Eastern Equine Encephalomyelitis (WEE - EEE)	The risk of contacting WEE will vary with the year. Annual vaccination is recommended. Two doses required annually to maintain good immunity.	1st dose - late spring 2nd dose - 2-4 weeks later	One dose annually in the spring
Venezuelan Equine Encephalomyelitis	No need to consider vaccination except in some southern States.		
Tetanus	The risk in Alberta is generally low but since the vaccine is conveniently available in combination with WEE-EEE vaccine, annual vaccination is recommended.	1st dose - anytime after 3 months of age 2nd dose - 4-8 weeks later	One dose (give in spring with WEE- EEE)
Influenza	The risk of getting "flu" is high if the horse is going to horse shows, trail rides, gymkhanas races, therefore these horses should be vaccinated. Probably not necessary to vaccinate horses that never leave home.	1st dose - 6-12 weeks prior to circuit, i.e. January, 2nd dose 4-12 weeks later	One dose early in spring
Equine Viral Rhinopneumonitis	The need for immunization is controversial because the virus is suspected of causing some "flulike" upper respiratory conditions at the race track but virus isolates have not been carried out. The virus has been isolated from abortions on some farms in Western Canada. Consult your veterinarian.	Same as influenza for race horses. Pregnant mare at 5, 7 and 9 months of pregnancy each year	One dose in Spring in race horses
Rabies	Recommended only in face of an outbreak.	One dose	One dose
Strangles	A vaccine may be recommended in the face of a very serious outbreak in a group of young horses, but the vaccines vary in their protective capabilities and in their inherent side effects. Consult your veterinarian.		

Figure 1. — Parts of a light horse



U - Unsoundness B - Blemish

Figure 2. — Common unsoundnesses and blemishes

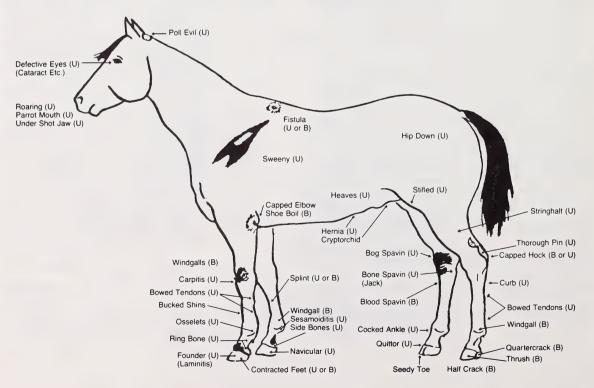
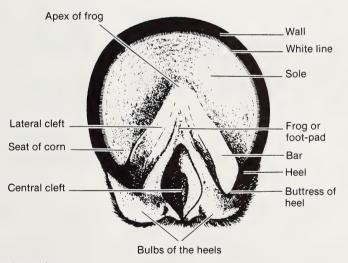


Figure 3. — The external foot or hoof

Toe

(From Horse Management, Edited by John Hickman 1984) Wall -Periople Parallel rings of horn



Heel

Quarter

Figure 4. — Front foot-bearing surface (From Horse Management, Edited by John Hickman 1984)

This section is included to give the reader a background concerning the recognition, cause, treatment and outcome of the common conditions which produce lameness in the horse. An attempt has been made to keep the information practical and in language the layman will understand. However it is necessary to refer to anatomical, conformational and physiological aspects of the horse in order to give accurate information. (Figure I and 2)

Many and varied treatments are available for lameness, once an accurate diagnosis is made. Diagnosis may be as simple as a physical examination done by your practising veterinarian or as complicated as special radiographic tests at a veterinary teaching hospital. Treatments vary from simple rest to complicated surgery, with corrective trimming and shoeing, anti-inflammatory drugs, bandaging, cold hosing, acupuncture, electrical stimulation and many others coming in between. It must be remembered that an accurate diagnosis is the first step in any case, and veterinary services should be used.

Most lameness, particularly of the forelimbs, is found from the fetlock down. In the hind limb, the hock is often involved. As in any case of lameness, start to look for causes from the foot up. See Figures 3 and 4 for diagrams showing the parts of the foot.

#### STONE IN THE FOOT

This occurs suddenly, and usually while at work, particularly on a gravel road. Stones often lodge between the shoe and the frog causing considerable discomfort. Stones should be removed immediately before further damage can occur.

# **BRUISED SOLE**

This is direct injury of the flat of the hoof or sole by stones, irregular ground or injury. Bad shoeing may also cause this. Rest and corrective shoeing are usually all that is needed. If unresponsive, veterinary assistance should be sought.

# **CORNS**

Corns are the result of bruising of the sole between the bar and the hoof wall. This is most often the result of poorly fitting shoes or neglect to have the horse re-shod at reasonable intervals. Corns can be simple and classified as dry, or they can become infected. Dry corns are treated by relieving the pressure. Remove the shoe, clean the foot thoroughly and reapply a ¾ length shoe, or rest without shoes if possible. If the corn is infected it will appear moist and be draining serum or pus. In these cases veterinary assistance should be obtained.

# PRICKED FOOT OR PUNCTURE WOUNDS OF THE FOOT

Puncture wounds of the foot are common and can cause severe lamenesses that may lead to permanent disability or death if not properly treated. As with any wound that allows bacteria to enter the body, there is a serious threat of infection occurring, necessitating veterinary care to prevent serious consequences (e.g., tetanus). Abscesses can develop in as little as two days following the initial injury. Often the site of puncture is very difficult to find by visual examination; a hoof knife, hoof testers and/or radiographs can be used to locate and evaluate the injury. The outcome of foot punctures depends on the area of the foot affected, the type of puncturing object, and whether secondary infection occurs. In all cases early veterinary care is essential to evaluate the damage done and prevent further damage from occurring.

#### HOOF CRACKS

Hoof cracks, extending from the ground surface upward, are common problems in horses' hooves which are not trimmed or are excessively dry. Injury to the hoof forming tissue at the coronary band can also result in cracks; these extend from the coronary band downwards. Severe lameness can be produced if bacteria enter the cracks and gain access to the sensitive structures of the foot causing infection. Cleaning and treating of the crack, followed by proper trimming and shoeing can be used to alleviate the condition until the hoof wall grows out (about 1 cm growth per month). The application of fibreglass or plastics to the hoof wall can also produce excellent results when applied by a competent farrier or veterinarian. Hoof cracks can usually be prevented by periodic hoof trimming and preventing the hooves from becoming excessively dry and hard. Standing a horse in water or mud periodically will help to keep its feet soft. The application of hoof dressing is helpful to prevent moisture escaping from the hoof; however, these should be applied to hooves already dry. Neat's foot oil is an excellent agent for moisturizing dry hooves.

# **THRUSH**

Thrush is a bacterial infection of the frog and sole of horses kept in unclean conditions. Affected feet will have a very offensive odor and will show a black discharge around the frog. Lameness will result if the condition is allowed to progress far enough to affect the sensitive structure of the foot. Proper cleaning and trimming of the foot along with proper stable sanitation will remove the cause of the infection. Affected horses can be treated by cleaning and trimming the foot and disinfecting it with a phenol or iodine

solution. The foot can also be packed with cotton soaked with 15 per cent sodium sulfapyridine solution.

#### LAMINITIS OR FOUNDER

Laminitis is an inflammation of the laminae that bind the hoof wall to the coffin bone. The causes of laminitis are varied and in many cases obscure. The commonly recognized causes are:

overeating of grain ingestion of cold water by a hot horse retained afterbirth overfat horses

idle horses pastured on lush green pasture (especially if clover or alfalfa are present)

Regardless of the cause, the signs of laminitis will be similar. In the early or acute stages the hoof and coronet will be hot, the horse will often sweat and will be reluctant to move. Because the front feet are usually the most severely affected, the horse will be camped out in front (front feet out in front of the body) and camped under (hind feet well under the body) behind in an attempt to remove weight from his front feet. In cases where all four feet are affected the horse will stand under himself both in front and behind as though he were attempting to balance on a barrel.

In advanced or chronic cases of founder there is usually an exaggeration of the ring formation on the hoof wall and there will be a decreased arch to the sole (dropped sole). In severe cases the coffin bone will protrude through the sole. The afflicted horse may spend most of his time lying down. A chronically foundered horse will exaggerate his attempts to land heel first while in motion.

Treatment of early laminitis varies with the cause. Whenever a horse is suspected of having laminitis a veterinarian should be called immediately and the horse exercised (mild exercise) until the veterinarian arrives.

If not severely affected, horses with chronic laminitis can be useful for most types of work if proper foot care is instituted.

#### **NAVICULAR DISEASE**

Navicular disease is lameness of the front feet caused by injury to the navicular bone. It is a very common lameness, particularly in breeds which have genetic defects in conformation. Defects in conformation such as upright pasterns and straight shoulders and/or small feet impair the shock absorbing mechanisms of the front legs and predispose the horse to navicular disease. Similarly, heavy use on hard ground will increase the probability of navicular disease. Affected horses are usually lame in both front feet. Because of the pain they will alternately "point" or advance one front foot at a time to remove the weight from it and thus reduce the pain. In the early stages of the disease, resting the horse can make the symptoms disappear, but when

returned to work the horse will soon become lame. Eventually, the affected foot or feet will become smaller than normal, the walls steeper, the heels contracted and the frog shrunken because the horse will attempt to reduce the pain by landing toe first rather than heel first as he moves. Corrective shoeing, pain killers and neurectomy (cutting the nerves to the back surface of the foot) can prolong the useful lifespan of the horse. Accurate diagnosis and treatment of navicular disease requires radiographic examination of the navicular bone in the horse's hoof, but at present there is no known cure for the condition.

#### **OSSELETS**

A form of arthritis of the fetlock which is usually the result of strain and repeated injury from hard training in young horses. There is usually pain and swelling over the fetlock and the horse moves with a short choppy gait. The condition is referred to as green osselets at the beginning. It progresses to a build up of bone around the joint. Some bone may break off to produce joint mice. When this occurs the horse will have repeated fetlock problems and surgery may be necessary to remove the fragment of bone in the joint, or joint mice. Early diagnosis, treatment and rest improve the prognosis for recovery.

#### WINDPUFFS

Windpuffs are fluid filled distentions of a joint or tendon. They are not accompanied by heat or pain. Usually windpuffs occur during hard work with young horses. Normally windpuffs themselves do not result in lameness and treatment is of little practical value. They are particularly common in the area of the fetlock joint. They can be prevented by reducing the strenuous work at too young an age.

# RINGBONE

Ringbone occurs following injury to the bone-forming tissue of the long or short pastern bones or the coffin bone. As a result there is a build up of bone around the injured area. If the enlargement occurs on or around the pastern joint, it is referred to as low ringbone. The cause of ringbone is usually injury predisposed by faulty conformation, but it can occur following wire cuts, or blows to the area below the fetlock joint. As the ringbone forms, the area above the coronet will be enlarged, hot and painful. The outcome of ringbone will depend on the severity of the condition and location, but invariably leads to impaired usefulness of the horse. Most cases of ringbone can be improved by proper veterinary and horseshoeing care.

# **SIDEBONES**

Side bones refer to ossification (turning to bone) of the lateral cartilages of the coffin bone. They are usually caused by poor conformation or improper shoeing resulting in excess trauma to the cartilage. Lameness does not necessarily accompany sidebones but stocking up of the lower limb often occurs owing to decreased efficiency of the blood pumping mechanism of the foot. They are especially common in the front feet and normally can be felt as a firmness in the area above the bulbs of the heels. Corrective trimming and shoeing by an experienced farrier is often helpful to relieve this condition.

#### **BOWED TENDON**

Bowed tendon is characterized by inflammation and tearing of the fibers of the flexor tendons at the back of the cannon bone. It is much more common in the front legs and can occur at any point from the knee to the attachment of the deep flexor tendon on the coffin bone. The cause of bowed tendons is most often an injury due to the severe strain of racing or hard work. Long weak pasterns, inadequate training, poor ground conditions, and improper shoeing or trimming predispose a horse to bowed tendons. Heat, swelling and pain will be present over the affected area in the early stages. Recovery from a bowed tendon depends on how severe the bow is, how soon proper veterinary treatment is initiated, whether the horse has a conformational predisposition toward bowing, and the use for which the horse is intended. In most cases, a long recovery period and intensive care is necessary to return the horse to a sound condition.

# **BUCKED SHINS**

Bucked shins are caused by stress applied to unfit or immature cannon bones. Affected horses will show pain, heat and swelling on the front surface of the cannon bone. One or both shins may be affected. Veterinary advice should be sought when the condition occurs. Although complete stall rest will allow the present buck to heal, it will halt the conditioning of the cannon bones and the shins may buck again when the horse is returned to work. Therefore, it is recommended that exercise be reduced but not ceased entirely until the injury has healed. Avoiding quick starts and hard running until a young horse is well conditioned will help to prevent the condition in the first place.

# **SPLINTS**

Splints are a condition found usually on the insideside of the front legs of young horses. They are the result of an inflammation of the ligament which binds the splint bone to the cannon bone or caused by a fracture of the splint bone. Usually, any lameness produced by splints is transient and will disappear as the excess fibrous tissue, formed by the inflammatory reaction, turns to bone. Lameness from splints is rare in horses over six years old.

In some cases however the splint may occur very close to the knee joint or under the suspensory ligament. In these cases the new bone growth may interfere with the action of the knee joint or cause irritation of the suspensory ligament resulting in permanent lameness. Heat, pain and swelling will be present over the area of the splint as it forms; as bony replacement occurs the splint will contract, become more firm and cool off. Poor conformation which places excess strain on one or the other side of the limb is a major cause of splints, although heavy training can cause splints in a well-formed young horse. Traumatic injury caused by blows to the area of the splint bones can also result in splints. Veterinary attention and rest should be instituted when a splint is suspected.

# **BOG SPAVIN**

Bog spavin is a fluid filled swelling of the hock joint caused by poor conformation, or an injury to the joint. Bog spavins usually do not produce lameness but cause swelling on the outside, just ahead of the point of the hock and on the front inside surface of the hock joint. When one of the swellings is squeezed often the other will enlarge and tighten. Veterinary treatment will be useful if the original cause of the bog spavin can be removed.

# **BONE SPAVIN (JACK)**

Bone spavin is a lameness caused by an abnormal increase of bone on the inside surface of the hock joint. Poor conformation, especially sickle and/or cow hocks, are predisposing factors to the development of this condition. It is often caused by the strain of work. Pain is produced when the hock is flexed and affected horses will show lameness of varying severity, often it is much less obvious after exercise. The condition is usually accompanied by a firm enlargement on the inside surface of the hock. When bone spavin is suspected, hold the suspected leg in a flexed position for about two minutes and then lead the horse away at a trot. If bone spavin is present the lameness will be greatly accentuated. The lameness can be relieved with the use of prescribed pain killers, corrective shoeing or by injecting the area with antiinflammatory agents.

# **CURB**

Curb is a sprain of a ligament causing a swelling just below the point of the hock. Poor conformation, such as sickle and/or cow hocks predispose the horse to curb. Violent straining or direct trauma are the most common causes of curb. Affected horses should be given early veterinary care followed by a long period of rest. If not caused by poor conformation, properly treated curbs should not affect the usefulness of the horse.

# **THOROUGHPIN**

This is a sprain with inflammation of the deep flexor tendon at the hock. A swelling just above the point of the hock is the result. The cause is usually a direct injury or overwork. A lameness may be present immediately after the injury, but usually disappears. In most cases, absolute rest is the recommended treatment. Veterinary treatment may be required.

# **CAPPED HOCK AND ELBOW**

Capped hock is a firm enlargement on the point of the hock caused by direct injury to the area. Capped elbow is an enlargement on the point of the elbow caused by direct chronic injury. This is often called shoe boil. Lameness is not usually produced but the enlargement can be unsightly and permanent unless reduced by veterinary care.

# **UPWARD FIXATION OF THE PATELLA**

This occurs when the patella (knee cap) locks over a ridge on the distal end of the femur. As a result both the stifle and hock are locked in an extended position.

Horses which are too straight (post-legged) in the hind legs are most commonly affected. There are variations in degrees of affliction. Often the stifle and hock will be locked in rigid extension with the horse being unable to release the patella, while in other cases intermittent locking of the stifle and hock will occur. If the horse is forced to walk while the patella is locked, it will drag the toe along the ground and pain is exhibited. In still other cases, the patella will "catch" enough to cause only an abnormality in movement. This will be more evident when the horse is circled toward the affected leg and is difficult to distinguish from stringhalt. Affected horses will usually become progressively worse because the ligaments which hold the patella in place are stretched each time the patella catches. There is a relatively simple surgical procedure that can be used to alleviate this condition and it should be done as soon as possible after recognition of the symptoms in order to prevent permanent damage to the stifle joint. Corrective shoeing is also beneficial in mild cases.

#### STRINGHALT

Stringhalt is a condition affecting the hindlimbs whereby the horse loses control of limb flexion. The limb will jerk upwards uncontrollably. This jerky flexion of the limb is especially evident when the horse is backed or turned. The cause is unknown but nervous involvement is suspected. Affected horses tend to get progressively worse despite occasional periods of improvement concurrent with increased environmental temperature and/or increased use of the horse. A relatively simple surgical technique has been developed that is sometimes effective in alleviating the symptoms.

# **SWEENEY**

Sweeney results from the shrinkage of the muscles on the shoulder blade, usually as a result of nerve damage. This condition was most common in draft horses, and caused by pressure of the collar over the nerve. Although nerve regeneration is possible it takes a long time and does not often occur.

#### WOBBLER

The wobbler syndrome is a condition caused by damage to the spinal cord in the neck region resulting in lack of coordination in the affected horse. The exact cause is unknown but there is a genetic predisposition which may be aggravated by an accident. Long-necked horses with large heads seem particularly prone to the condition. Affected horses will show uncoordinated movement which is especially apparent in the hind legs and when the horse is turned in a tight circle. Such horses are not safe to ride. Because of the genetic predisposition they make undesirable breeding stock. Surgical stabilization of the spinal cord in the neck has been helpful in certain cases.

# **EPIPHYSITIS**

Epiphysitis is an inflammation of the growth plates of long bones at the bottom of the radius above the knee or the bottom of the cannon bone above the fetlock.

The cause is usually a combination of factors: over-feeding, mineral deficiency or imbalance, injury, conformation defect and excessive or inadequate exercise. Typically the problem is noticed in rapidly growing Quarter Horse or Thoroughbred foals that are fed a high grain ration which is improperly balanced.

Treatment includes rest and a ration formulation to correct mineral deficiencies, excesses or imbalances, and to restrict the rate of growth to a level compatible with normal skeletal development.

# TYING-UP SYNDROME (AZOTURIA, BLACK WATER DISEASE OR MONDAY MORNING DISEASE):

Tying-up usually occurs with race horses or jumpers that are being exercised regularly and fed well. Often a horse that has just been raced or exercised will show evidence of muscle pain and a short stilted stride. He then becomes very reluctant to move. Sweating usually occurs. The muscles in the hindquarters become tense, painful and may be swollen.

Azoturia or Monday Morning Disease is more severe and is most often noticed in heavily fed and worked horses that have been given a day or so of rest. Shortly after the horse has started working, it begins to show muscle stiffness, tremors and pain; and finally is unable to move. The urine may be reddish brown or black. A prolonged recovery may be necessary and horses are susceptible to more attacks. Kidney damage and muscle atrophy may occur with Azoturia but rarely with tying-up syndrome.

Prevention involves carefully regulating the diet, exercise and cooling off activities and reducing grain intake on days when horses are resting and in the case of racehorses, before a race. No permanent damage is likely to occur from mild attacks but the horse will be more susceptible to future attacks.

#### **FEEDING PROBLEMS**

#### **MALNUTRITION**

Malnutrition and starvation are serious man-made problems in horses and are especially prevalent in young horses during the winter.

Starvation occurs when horses are fed a diet insufficient in either quantity or quality over an extended period of time. It may be aggravated by parasite infestation, poor teeth, and/or competition from other horses. The results are gradual loss of stored energy followed by resorption of body fat and muscle. Muscular power and activity decrease with a loss of body weight that may reach as high as 50 per cent. There will be a decreased heart rate, blood pressure, mental depression, lack of sexual desire and increased susceptibility to infection. Death will eventually result caused by energy deficiency.

Starvation occurs much faster in young animals and those trying to survive during cold weather. Even a short period of nutritional restriction can stop skeletal growth and permanently stunt young horses.

Starvation is most inhumane, and guilty owners may be subject to prosecution. It can be easily prevented by feeding proper diets, feeding young horses separately from older horses, providing shelter during the winter, proper deworming and routine evaluation of the condition of horses. It should also become routine to have hay analyzed and the feeding program of the horse balanced every winter.

# COLIC

Colic simply means that the horse has abdominal pain. Generally there is distension of the stomach or intestine caused by an accumulation of feed, gas and/or fluid. The normal movements of the intestine may be static or over active (spasms). The horse with colic may have acute abdominal pain, kick at the belly, sweat, roll, sit and have increased pulse and respiration rates. The horse may be dehydrated, pass very little feces and in very severe cases

may vomit. As colics can be serious, a veterinarian should be called immediately. Delays can prove very costly. It is usually recommended that one keep the horse up, blanketed and walking until the veterinarian arrives to prevent the horse from injuring itself. Feed should be withheld, but water should be offered. Pay particular attention to see if the horse urinates or passes manure.

# **PARASITIC**

Parasites are responsible for most cases of colic in horses by obstructing and damaging the intestines. Blood worms often cause damage to intestinal arteries which result in reduced blood flow to segments of the intestine. The reduced blood flow causes a weakening of a section of the intestinal wall. This may become distended and either reduce passage of ingesta or rupture into the abdominal cavity. Also, large numbers of worms, especially roundworms, may cause obstruction in the intestine. Parasitic colcs are prevented by minimizing the worm burden.

#### IMPACTION

Impaction or blockage of the large intestine is usually caused by feeding low quality feed such as straw or poor hay. The feed remains in the stomach, cecum or large intestine for a long period of time as rate of microbial fermentation is low (especially if protein is deficient). If an animal is stressed because of cold weather or heavy work, it may eat more than is being digested and simply plug up. A shortage of water, eating snow or feeds that are diuretic (alfalfa) may cause the food to be too dry and cause blockage. Bad teeth, inadequate exercise or reduced intestinal motility may also cause blockage. Parasites (strongyles) may affect the blood supply to the intestine thus reducing motility and absorption which leads to colic. A veterinarian may, by rectal palpation, locate the blockage and assist in moving the ingesta. Surgery is often necessary but the

chances of success are low. A severe type of impaction occurs in newborn foals that retain their meconium (first manure). Veterinary attention is usually required and these cases sometimes require surgery.

# **GAS (FLATULENT)**

Although all feeds produce gas while being digested in the large intestine, some feeds produce more than others. For example, high quality alfalfa is fermented faster than most roughages and the excess gas may accumulate in the intestines causing pain and reduced motility. The increased pressure may also cause a buildup of harmful microflora (e.g. production of toxins from anaerobic organisms). A veterinarian may treat flatulent colics by increasing gut motility or by reducing gas production through medication.

# **ENGORGEMENT OF GRAIN**

Although horses can be fed large amounts of grain, they have to adapt slowly to a grain diet over a two to three week period. Engorgement of grain, especially barley and wheat, causes an increased production of lactic acid by microorganisms in the large intestine. This causes the intestine to draw in more fluid and may cause a reduction in motility as well. Besides colic, laminitis is also a threat with overeating of grain. The acidity may cause damage to the intestinal wall, resulting in the release of histamine which may cause laminitis. Ten pounds of wheat will usually cause engorgement and/or laminitis in an unadapted horse.

Veterinary treatment will usually include the use of medication to reduce the acidity and fermentation, increase gut motility and to help prevent laminitis.

#### **SPASMODIC**

Excitement, cold water, thunderstorms, moldy feed, a sudden change of feed, or parasites may cause increased peristalsis or spasms in the intestine which in turn may cause abdominal pain the horse. A veterinarian will use medication to reduce the excessive movement.

#### TWISTED INTESTINE

For reasons which usually defy explanation, a horse's small intestine may develop a twist thus causing a stoppage in movement of food and a buildup of fluid. A twist is extremely serious because of the risk of intestinal rupture and because of the rapid deterioration of the intestinal wall. Surgery is the only effective therapy available.

# **POISONING**

When the cause of sudden death is not readily known, owners often suspect poisoning. Actually death caused by poisoning is relatively uncommon.

Signs of poison affecting the gastrointestinal system may be diarrhea, salivation and colic. Signs of an affected nervous system include hyperexcitability, muscular twitching, uncoordination, paralysis, coma, convulsions, and abnormal movements and stance. When the liver's function is affected, the buildup of waste products may affect temperament and personality, and white areas of the body may become light sensitive. Other general signs of poisoning are a lack of appetite, dehydration and depression. Some poisons may cause a lack of oxygen or heart failure resulting in death.

In all cases careful veterinary examination is required because an accurate diagnosis is necessary.

#### POISONOUS PLANTS

Generally, horses will not consume poisonous plants unless forced to by a lack of forage or if they are mixed with other feeds. If some minerals are deficient in the diet horses may eat unusual things. Care should be taken when turning horses into new pastures, and in purchasing hay or grain. In most cases, large quantities of a poisonous plant are necessary to cause problems. Some types of poisonous plants are bracken fern, lupines, locoweed, water hemlock, Astragalus, Lathyrus and yellow star thistle. Ergot in grain is poisonous at levels greater than one kernel per 1000. Rye commonly has ergot in it. Foxtail is not poisonous but can cause considerable mechanical injury.

Two common plants which can be extremely poisonous to horses are alsike clover and sweet-clover hay. Alsike clover causes damage to the horse's liver, although it is relatively safe in cattle. Horses can show photosensitization or sunburn of white areas, or sudden death. With sweet-clover hay, a toxin called dicoumarin can build up. The dicoumarin buildup is a result of mold formation in poorly made sweet-clover hay. This is the active ingredient in warfarin rodent bait and causes uncontrolled and excessive bleeding. Early veterinary treatment is required.

# **NITRATE POISONING**

Common forage plants can cause livestock poisoning. Oat hay or straw is the most common cause of nitrate poisoning. Barley and wheat straw, corn, flax, sorghum and other forages, weeds, chiefly lamb's-quarters, have been found to contain toxic quantities of nitrate. Using high levels of nitrogen fertilizers may cause high nitrate levels in common grasses.

#### Conditions of Poisoning

Nitrate poisoning is most common when feed, particularly immature oats, has suffered drought late in the growing season. Forage crops from areas such as feedlots and cattle yards, where manure has accumulated for long periods of time, may be dangerous. Many common weeds can cause nitrate poisoning. Lamb's-quarters is particularly dangerous and should never be feed to livestock. Hay from shady areas on high-nitrogen soils may be dangerous, as nitrate accumulation increases with increased shading. Animals on poor feed are more susceptible to nitrate poisoning. This susceptibility can be reduced by including concentrates with a high carbohydrate content in the ration. However, this treatment may be precluded by the fact that animals usually prefer feeds high in nitrates, and unless care in management is exercised the animals will still ingest excessive amounts of the high nitrate feed.

# **Toxic Principle**

When plants with a toxic amount of nitrate are consumed by livestock, a conversion of nitrate to nitrite takes place in the intestinal tract. This nitrite is absorbed into the

bloodstream, where it combines with the hemoglobin, the oxygen carrier in the blood, to form methemoglobin. Methemoglobin does not combine with oxygen and when large quantities of it are present, the oxygen-carrying capacity of the blood is reduced to the extent that the animal dies of anoxia, which is similar to carbon monoxide poisoning.

Plants can be tested for nitrate content at provincial veterinary laboratories or all feed testing laboratories. Although the upper limit of safety for the horse has not been determined, forages with up to 1 per cent nitrogen in the form of nitrate-N are considered safe.

# POISONOUS CHEMICALS

Chemical poisoning is more common than plant poisoning and avoiding it deserves considerable attention. Some common sources are paint, used oil, batteries, weed sprays, insecticides and wood preservatives. Tables 3 and 4 list some common poisonous compounds.

Table 3. — Poisonings Characterized by Gastrointestinal Disorder

Poison	Source	Toxic Level	Other Signs	Diagnosis And Treatment
Arsenic	Insecticides Herbicides Rodenticides Wood preservatives	Oral, acute - 26-44 mg/kg Chronic: 1-3 g per day for 14 weeks to kill	Possibly, none in acute. Chronic - emanciation, in horses and mules. A marked resistance to post-mortem decomposition often is seen in arsenic poisoning.	Remove the source and give both symptomatic and supportive treatment. BAL has limited use in the horse. Reinsch test confirmed by marsh test plus a high arsenic content of liver, blood, urine, and kidney tissue.
Lead (The most toxic are more soluble)	Paints Insecticides Medicaments Fallout from smelters	Oral, acute - 11-44 mg/kg Chronic: as low as 55 mg/kg, if repeated over a long period	Dyspnea with roaring. A central nervous disturbance similar to encephalomyelitis or rabies (paralysis of lips). Stiffness of joints. Lead sulfide line on gums. Weakness.	Use 2% saline-sugar sol. of Ca- EDTA, intravenously at rate of 60- 80 drops/minute q.s. 1 g/13.5 kg liveweight, once daily for 3 days, skip 4 days, repeat. Peracute and acute poisoning kill before one can diagnose or treat the condition.
Selenium (Alkali disease)	Forage or grain on seleni-ferous soils	MLD is 3.3 mg/kg as sodium selenite equi- valent. Cumulative	Loss of hair from mane and tail; softened and abnormal hoof; lis- tlessness and emanciation; lesions in the kidney, liver, heart and spleen.	Remove from exposure; give sup- portive and symptomatic treatment and sharply improve the quality of the feeds (increase the sulfur-bearing proteins). Many severely poisoned animals never recover.
Organic Phosphates	Field and orchard insecticides Miticides	From less than 2.2 mg/kg	Horses have colic, general mus- cular weakness, miosis, perspi- ration and occasionally, profuse salivation.	Atropine to effect and 2-PAM at the rate of 22 mg/kg, i.v. repeating as necessary each 2 hours until the intoxication is controlled.
Phosphorus and zinc phosphate	Fireworks Rodenticides	2.2 mg/kg for yellow phosphorus 22-44 mg/kg for zinc phosphide	Violent colic, convulsions, garlic odor on breath. With zinc phosphine (PH <sub>3</sub> ) odor upon opening the stomach. Intensive petechial hemorrhages of serosa of bowel.	Symptomatic and supportive; use of mucilaginous liquids; universal antidote. Treatment is not very effective.
Phenol (Creosote)	Wood preservatives	Acute: 30 g is lethal Chronic: as little as .22 mg/kg as a persistent contact irritant	Acute: depression, ataxia, convulsions, paralysis, and terminal muscular tremors. Chronic: contact burns, and slower changes with encephalitis.	Remove from exposure and give both symptomatic and supportive treatment. Treat the burns according to methods useful for any chemical burn.

Table 4. — Poisonings Characterized by Nervous Manifestations

Agent	Source	Toxic Level	Chief Signs	Diagnosis And Treatment		
Chlorinated hydrocarbons	Insecticides (dips, sprays) Field and tree pesticides	Variable and may be acute or chronic. From 33 mg/kg to 6167 mg/kg	Convulsions and colic, intermittent in type.	History of exposure. Sedate to effect. Symptomatic and supportive therapy. Chloral hydratemagnesium sulfate or barbiturates to control the C.N.S. disturbance		
Fluoro-acetate	Rodenticides	11 mg/kg kills in a few hours.	Intermittent convulsions, colic, possibly diarrhea.	History of rodent eradication campaigns, plus a typical and rapid course. There is no effective antidote for horses.		
Lead	Paints	As low as 55 mg/kg if constant intake of lead.	Convulsions. Recurrent laryngeal nerve paralysis (roaring).	Use Ca-EDTA and supportive treatment. Diagnosis by demonstration of lead in toxic amounts of analysis of blood, liver, kidney, urine; Hammond test.		
Organic phosphates	Pesticides	From less than 2.2 mg/kg to more than 2202 mg/kg	Terminal-type convulsions.	Atropine and 2-PAM. Diagnosis depends on history of exposure plus the severe colic and signs of parasympathetic stimulation.		
Strychnine	Pesticides	2.2-4.4 mg/kg	Typical convulsions.	History of exposure plus easily-induced convulsion. Control convulsions with chloral.		
Thallium	Rodenticides	30.8 mg/kg	Tremors, dysphagia, depression, paralysis.	History of exposure. Intense hemorrhagic gastroenteritis, hepatitis, nephritis, and hyperemia of brain. Hammond test on urine. Only antidote of promise is dephenylthiocarbazone at 77 mg/kg tid. Not very practical for horses.		
Antu	Rodenticides	26 mg/kg to kill in 8 hours at first dose.	Severe depression, death without convulsion.	No effective antidote, but sublethal and repeated exposure results in development of a tolerance which exceeds 4 times the initial lethal dose.		

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